U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 28 September 2004

CASE NO. 2003-BLA-5873

In the Matter of:

MARYLYN L. CLOWSER,
Survivor of DONALD E. CLOWSER,
Claimant

v.

PERRYSVILLE COAL COMPANY,
Employer

and

DIRECTOR, OFFICE OF WORKERS
COMPENSATION PROGRAMS,
Party-in-Interest

APPEARANCES:

Debra L. Henry, Esquire
For the Claimant

John J. Bagnato, Esquire
For the Employer

John M. Strawn, Esquire
For the Director

Before: RICHARD A. MORGAN
Administrative Law Judge

DECISION AND ORDER - DENYING BENEFITS

This proceeding arises from a claim for benefits filed by Marilyn L. Clowser, the surviving spouse of Donald E. Clowser, a now deceased coal miner, under the Black Lung Benefits Act, 30 U.S.C. §901, *et seq*. Regulations implementing the Act have been published by the Secretary of Labor in Title 20 of the Code of Federal Regulations. [1]

[1] The Secretary of Labor adopted amendments to the "Regulations Implementing the Federal Coal Mine Health and Safety Act of 1969" as set forth in Federal Register/Vol. 65, No. 245 Wednesday, December 20, 2000. The revised

Black lung benefits are awarded to coal miners who are totally disabled by pneumoconiosis caused by inhalation of harmful dust in the course of coal mine employment and to the surviving dependents of coal miners whose death was caused by pneumoconiosis. Coal workers' pneumoconiosis is commonly known as black lung disease.

A formal hearing was held before the undersigned on January 14, 2004, in Pittsburgh, Pennsylvania. At that time, all parties were afforded full opportunity to present evidence and argument as provided in the Act and the regulations issued thereunder. Furthermore, I held the record open, in order to allow the submission of the post-hearing deposition of Dr. Green and closing arguments (TR 8, 48-49). The transcript of Dr. Green's deposition testimony has been marked and received in evidence as Claimant's Exhibit 4 (CX 4). By Order Granting Request for Extension of Time to File Closing Arguments, dated March 5, 2004, and Order Granting Simultaneous Submission of Closings, dated March 16, 2004, I extended the deadline for submission of the parties' closing arguments until April 26, 2004, and ordered that the submissions be made simultaneously.

At the close of the hearing, Employer's counsel withdrew Dr. Bush's supplemental report, dated August 6, 2002 (DX 20). Accordingly, Director's Exhibit 20 is not in evidence. On the other hand, Dr. Bush's initial report, dated June 5, 2002, which is limited to autopsy findings, remained in evidence (DX 19). Furthermore, I ruled that the admission of Dr. Bush's deposition transcript is limited to those portions of his testimony relating to the pathology report (EX 4; TR 47-48). Having considered the parties' closing arguments regarding this issue, I reiterate my prior ruling. Furthermore, I find that the (partial) admission of Dr. Bush's deposition transcript is inconsequential, since the admitted portions of the deposition simply reiterate Dr. Bush's findings as set forth in his pathology report, dated June 5, 2002 (*Compare* DX 19 & EX 4, pathology analysis).

In summary, the record consists of the hearing transcript, Director's Exhibits 1 through 40, except DX 20 (DX 1-19, 21-40), Claimant's Exhibits 1 through 4 (CX 1-4), and Employer's Exhibits 1 through 6 (EX 1-6). However, Employer's Exhibit 4 is limited to Dr. Bush's deposition testimony regarding the pathology evidence.

The findings of fact and conclusions of law which follow are based upon my analysis of the entire record, including all documentary evidence admitted and arguments made. Where pertinent, I have made credibility determinations concerning the evidence.

Procedural History

On October 24, 2001, Donald E. Clowser passed away (DX 11). On January 28, 2002, the Claimant, Marilyn L. Clowser, filed the current application for black lung benefits under the Act, as his surviving spouse (DX 2). This claim was denied by the District Director in a Proposed Decision and Order, dated January 16, 2003 (DX 30). Following Claimant's timely request for a formal hearing (DX 33), this matter was referred to the Office of Administrative Law Judges in May 2003 for *de novo* adjudication (DX 38-40). I was assigned the case on

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Part 718 regulations became effective on January 19, 2001. Since the current claim was filed on January 28, 2002 (DX 2), the new regulations are applicable (DX 40).

September 11, 2003. A formal hearing was held on January 14, 2004, and the record was held open until April 26, 2004. [2]

Issues

I. Whether the person whose death the claim is based on is a miner?
II. Whether the miner worked as a miner after December 31, 1969?
III. Whether the miner worked at least 15 years in or around one or more coal mines?
IV. Whether the miner's pneumoconiosis arose out of coal mine employment?
V. Whether the miner's death was due to pneumoconiosis?
VI. Whether the named Employer is the Responsible Operator?

(DX 38; TR 5-7).

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Background and Employment History

A. Coal Miner, Post-1969, and Length of Coal Mine Employment

On the application for benefits form, signed by Claimant on January 26, 2002, she alleged that her deceased husband had engaged in coal mine employment for 15 years (DX 2). On that same date, Claimant signed an Employment History form, in which she listed various coal mine and non-coal mine jobs. The Employment History form stated, in pertinent part:

Employer	Industry	Occupation	Employment Period
P&N Coal	Strip Mining	Heavy equipment operator	10/64 – 12/64
F.E. Cable Trucking	Coal Transportation	Truck Driver	3/70 – 2/71
Gold Builders	Strip Mining	Heavy equipment operator	8/71 – 6/79
Armitrage Trucking	Coal Transportation	Truck Driver	8/79 – 9/81
Perrysville Coal	Coal Transportation	Truck Driver	10/81 – 8/85

(EX 4)

The record also contains a "Description of Coal Mine Work and Other Employment" form, which was received by the District Director's office on the same date as the Employment History form (DX 5; *Compare* DX 4). The description of Mr. Clowser's duties while working for Employer is listed as follows:

[2] Due to an apparent clerical and/or mailing error, the transcript of Dr. Green's deposition, dated February 11, 2003, was not received by this Office until September 1, 2004. Furthermore, the following day, Claimant's counsel sent a corrected page 22 to this Office, which has been made a part of Claimant's Exhibit 4 (CX 4). Accordingly, the record was re-opened to allow the belated receipt of Dr. Green's deposition transcript. I note, however, that no prejudice has been caused by such action. The closing arguments of Employer and Claimant, which were filed on April 22, 2004 and April 23, 2004, respectively, both made specific references to Dr. Green's deposition. The Director's closing brief, dated April 26, 2004, focused solely on the responsible operator issue.

> Driving truck for Perrysville Coal Company/Calvin McElroy (Owner) 610 Sportsman Drive Apollo PA 15613 hauling coal from cleaning plant at Canterbury Coal R D 1 Avonmore PA 15618.

(EX 5). In addition, the following other coal mine jobs were listed:

Truck Driver	August 1979 - September 1981
Heavy Equipment Operator	August 1971 – June 1979
Truck Driver	March 1970 – February 1971

(EX 5).

Employer cites Director's Exhibit 5 (*i.e.,* "hauling coal *from* cleaning plant" language) and contends that this indicates that Mr. Clowser "was hauling prepared coal from the cleaning plant to consumers (Employer's Closing Argument, p. 2). However, as stated in the closing arguments presented by the Claimant and Director, respectively, Director's Exhibit 5 is unsigned and the credible testimony of Claimant and Sherri Umbaugh, who prepared the document, establishes that Mr. Clowser actually hauled coal from the mine to the cleaning plant (TR 16-20, 35-37; *See also* Claimant's Closing Argument, p. 2; Director's Closing Argument, p. 3). Although an affidavit by Robert Brunermer, a principle of Kenneth R. Sloan Insurance Agency, states that Employer was a coal trucking operation involving hauling of clean coal to users and consumers (DX 22), Claimant provided a written statement to the contrary (DX 22). Moreover, Richard L. Boarts who had also worked for Employer provided a written statement supporting Claimant's position, and expressly stating that they hauled unprocessed coal from the mine to the cleaning plant (DX 25).

Having carefully considered the conflicting evidence, I find that Claimant has met her burden of establishing that Mr. Clowser's work for Employer involved hauling unprocessed coal from the mine to the cleaning plant. Therefore, I find that Mr. Clowser's work for Employer constituted coal mine work. Accordingly, Claimant has established that Mr. Clowser was a coal miner, that her husband worked as a miner after December 31, 1969, and that Employer is the properly designated responsible operator.

The length of coal mine employment issue is more problematic, since Claimant's testimony, as well as that of Ms. Umbaugh, were ambiguous regarding the status of the coal (*i.e.,* raw versus processed) in the other coal transportation (*i.e.,* trucking) jobs. (TR 28-31, 37-39). However, the Social Security records confirm that, in addition to Mr. Clowser's coal mine work for Employer, he worked four quarters for P & N Coal Company in 1964-65. Furthermore, the Social Security records also confirm that Mr. Clowser worked at Gold Builders & Contractors for about 8.5 years. Moreover, Mr. Clowser worked for Employer a period of about 4 years (DX 8).

In view of the foregoing, I find that Claimant has established approximately 13.5 years of coal mine employment. Moreover, any discrepancy between my finding and the 15 years of coal mine work alleged by Claimant is inconsequential for the purpose of rendering a decision herein.

B Date of Filing

Claimant, Marilyn L. Clowser, filed her claim for survivor's benefits under the Act, on January 28, 2002 (DX 2). Employer has stipulated, and I find, that the claim was timely filed (DX 38).

C Responsible Operator

Since the miner's last coal mine employment of at least one calendar year was with the Employer, Perrysville Coal Company, I find that it is the properly designated responsible operator (DX 2, 4, 7, 8).

D. Dependents

Claimant, Marilyn L. Clowser, has no dependents for purposes of augmentation of benefits under the Act (DX 2).

E. Personal, Employment, and Smoking History

The former miner, Donald E. Clowser, was born on July 17, 1926. He married Marilyn L. Clowser on January 24, 1959. They remained married until the miner's death on October 24, 2001 (DX 2, 9, 11; TR 10).

Claimant stated that her husband engaged in coal mine employment for approximately 15 years ending in August 1985, when there was a lockout (DX 2; TR 12). The Social Security records indicate that Mr. Clowser worked sporadically in 1986-88, and do not reflect any earnings thereafter (DX 8).

Claimant testified that, when her husband left coal mining, he had difficulty walking and couldn't do things like he could before, such as mowing the lawn. Claimant also noted that Mr. Clowser felt better when he was doing things rather than simply sit around and that he enjoyed working with his hands on small engines and things (TR 12-13). Claimant stated that her husband had trouble sleeping. He raised the head of the bed and/or slept in a recliner, because he had trouble breathing when lying flat (TR 13-14).

Claimant acknowledged that her husband used to smoke about one pack of cigarettes per day. She initially stated that he quit smoking in about 1984. However, Claimant later testified that her husband quit smoking at or around the time that they moved into their new house, which was about "12 years" ago (*i.e.*, January 1992). Furthermore, Claimant did not specify the length of Mr. Clowser's smoking history. However, the medical evidence clearly establishes that Mr. Clowser had a significant cigarette smoking history. For example, the History and Physical Examination report, dated October 4, 2001, by Dr. Syed N. Husaini, states that Mr. Clowser is a "former smoker" who smoked "one pack per day for at least 45 years…(ending)…in 1994." (DX 17). On October 5, 2001, Dr. John Lister, a consulting physician, reported that Mr. Clowser "was a pack to pack and a half day smoker for approximately thirty five years." (DX 17).

Claimant testified that, in 1995, her husband was hospitalized for pneumonia. Thereafter, Mr. Clowser was not hospitalized until the "last event" when he was admitted to undergo a heart catheterization. However, Mr. Clowser never underwent the catheterization, because he had a blood clotting problem (TR 15).

Medical Evidence

The parties stipulated, and I find, that the evidence establishes the existence of (simple) pneumoconiosis (DX 38; TR 6). Furthermore, "total disability" is not a relevant issue, since this is a survivor's claim. The crux of this case rests on the disposition of the "death due to pneumoconiosis" issue.

The case file contains extensive medical records which confirm the diagnosis of pneumoconiosis and refer to Mr. Clowser's shortness of breath and COPD among various numerous other conditions (DX 13, 14, 15, 16, 17). Furthermore, Dr. Paul J. Brayer, who treated Mr. Clowser for many years at Valley Family Medicine, issued a report, dated April 24, 2002, stated:

> Mr. Clowser had multiple medical problems prior to his death including, hypertension, coronary artery disease, and chronic lung disease consistent with black lung. In recent years his lung disease was severe and disabling requiring treatment with oral block dilators, inhale block dilators, oral steroids and inhaled steroids. In spite of this very aggressive therapy the patient remained chronically short of breath with a significant on-going cough. Findings at autopsy…confirmed the presence of black lung.

(DX 18)

For the purpose of rendering a decision herein, however, the more relevant evidence consists of the following: the miner's death certificate (DX 11), the autopsy report co-authored by Drs. Omalu and Wecht (DX 12), and the medical opinions of Drs. Bush (DX 19; EX 4), Hurwitz (EX 1, 6), Fino (EX 2, 5), Green (CX 1-4), and Tomashefski (EX 3).

The miner's death certificate (DX 11), which was signed by Dr. Paul Brayer, one of Mr. Clowser's treating physicians (DX 13, 18), states that Mr. Clowser died on October 24, 2001. The immediate cause of death was reported as "Cardiopulmonary Arrest" due to "Coronary Artery Disease." No other conditions were listed as contributing causes of the miner's death (DX 11).

The autopsy report, dated October 25, 2001, was co-signed by Dr. Bennet I. Omalu and Cyril H. Wecht (DX 12). Dr. Omalu is listed as the "Forensic Pathologist, Prosector" and Dr. Wecht is described as the "Forensic Pathologist (who) has reviewed the autopsy materials and this protocol (DX 12, p. 3). Dr. Omalu's credentials are not in evidence, except for the notation that he is a Forensic Pathologist. On the other hand, Dr. Wecht's lengthy *curriculum vitae* is in evidence. It reveals that Dr. Wecht specializes in Anatomic, Clinical and Forensic Pathology, and Legal Medicine and that he has published extensively (DX 12). The autopsy report is 14+ pages in length. However, the vast majority of the report is wholly unrelated to the miner's

cardiopulmonary system. Furthermore, Dr. Wecht's "microscopic examination" analysis, as set forth therein, was minimal. It simply stated: "The microscopic examination is consistent with the gross autopsy observations. The histological findings are reflected in the final pathological diagnoses." (DX 12). In summary, Drs. Omalu and Wecht reported the following:

> FINAL PATHOLOGICAL DIAGNOSES:
>
> Arteriosclerotic Cardiovascular Disease:
> Myocardial infarction, acute and subacute, focal.
> Myocardial fibrosis, interstitial, microscopic.
> Atherosclerosis of left coronary artery, mainstem, severe.
> Atherosclerosis of left anterior descending coronary artery, severe.
> Atherosclerosis of right coronary artery, moderate.
> Atherosclerosis of left circumflex coronary artery, mild.
> Atherosclerosis of aorta, mild.
>
> Chronic Obstructive Pulmonary Disease:
> Pulmonary emphysema, bilateral.
> Pulmonary interstitial pneumonitis, bilateral.
> Granulomatous pneumonitis, bilateral, NOS.
> Pulmonary interstitial fibrosis, bilateral.
> Fibrohyaline and fibroanthracotic macules and micronodules.
> Pulmonary interstitial anthracosis, bilateral.
> Pulmonary hypertensive vasculopathy.
> Fibroanthracosis of peribronchial lymph nodes.
> Fibrous pleuritis.
>
> Pulmonary edema and congestion
>
> Splenomegaly, congestive.

(DX 12).

In summary, the autopsy report clearly indicates that Mr. Clowser suffered from numerous medical problems which primarily involved his heart and lungs. Furthermore, the diagnosis of "pulmonary interstitial anthracosis," which is listed among the numerous diagnoses, supports the finding of pneumoconiosis. However, the autopsy report fails to specifically address the death due to pneumoconiosis issue (DX 12).

Dr. Stephen T. Bush, who is Board-certified in Anatomic and Clinical Pathology and in Medical Microbiology (EX 4, Deposition Exhibit 2), issued a report, dated June 5, 2002, which primarily focused on pathology findings (DX 19). Following his analysis of the histologic slides, Dr. Bush concluded:

> The histologic findings in conjunction with the gross examination of the lungs show that Mr. Clowser had a very mild degree of simple coal worker's pneumoconiosis. This degree and extent of occupational disease was too limited to have caused or contributed to death. Death appears to have occurred from an acute myocardial infarction due to atherosclerotic coronary artery disease. Coal mine dust exposure does not cause or contribute to the development [of] coronary artery disease leading to death from myocardial infarction.

(DX 19). Dr. Bush reiterated the foregoing opinion in his deposition testimony on July 23, 2003 (EX 4). [3]

Dr. Larry E. Hurwitz, who is Board-certified in Internal Medicine and Cardiovascular Disease, issued a report, dated September 3, 2002, in which he discussed and analyzed various medical data (EX 1). Dr. Hurwitz reported that the autopsy findings demonstrated evidence of "acute and sub-acute myocardial infarction in the setting of severe coronary artery disease. The description of the heart indicated normal weight. The left ventricle was hypertrophied measuring 1.5cm (up to 1.1 cm normal). The right ventricle was described as unremarkable and of normal chamber size. The right ventricle measured 0.4cm. The autopsy report also identified the presence of chronic obstructive pulmonary disease." Furthermore, Dr. Hurwitz listed the multiple disease processes which had been set forth on the autopsy report under the heading of chronic obstructive pulmonary disease. Dr. Hurwitz also that Claimant had evidence of significant coronary and myocardial disease. In his report, Dr Hurwitz included information regarding an echocardiogram, dated May 18, 1999, which demonstrated severe left ventricular dysfunction and moderately severe pulmonary hypertension; a stress echocardiopathy, dated October 2, 2001, to evaluate exertional chest pain; and the cancellation of a scheduled cardiac catheterization due to a finding of progressive thrombocytopenia. Dr. Hurwitz also noted that Mr. Clowser's was last seen by his primary physician on October 23, 2001, when he reportedly was "doing okay," and received a flu shot. Mr. Clowser died the following day. Dr. Hurwitz also reported that a pulmonary function study, dated May 18, 2001, demonstrated obstructive and restrictive pulmonary disease, and was interpreted by a pulmonologist as demonstrating moderately severe emphysema. In conclusion, Dr. Hurwitz stated:

> It is my opinion, based on the records reviewed, that Mr. Clowser died as a result of an acute myocardial infarction in the setting of severe coronary and myocardial disease. Even though it is clear Mr. Clowser had significant pulmonary disease there is no evidence that the pulmonary disease was a factor in the events of the acute myocardial infarction.

[3] As outlined above, only Dr. Bush's deposition testimony regarding the pathology evidence is in evidence, because of the evidentiary limitations set forth in the new regulations (TR 47). Furthermore, Dr. Bush's report, dated August 6, 2002, was withdrawn at the formal hearing (TR 47-48). Similarly, the duplicate copy of Dr. Bush's report, dated August 6, 2002, which was submitted as a deposition exhibit (EX 4; Deposition Exhibit 3), is excluded from the record.

> It is my opinion with reasonable medical certainty that Mr. Clowser's occupational exposure to respirable coal dust was not a factor in the development of underlying coronary artery disease or in the precipitation of the acute myocardial infarction.

(EX 1).

In deposition testimony on November 6, 2003 (EX 6), Dr. Hurwitz stated that both he and Dr. Green had reported the result of a May 18, 1999 echocardiogram as demonstrating pulmonary hypertension, but that the echocardiogram was that of a patient other than Mr. Clowser (EX 6, p. 13). However, Dr. Hurwitz testified that he did not specifically rely on the echocardiogram, dated May 18, 1999, in reaching his ultimate conclusion, because he found nothing in the pathologic record that indicates Mr. Clowser had pulmonary hypertension (EX 6, p. 17). Dr. Hurwitz also questioned Dr. Green's finding of cor pulmonale. Dr. Hurwitz noted the differing measurements of the right ventricle by Dr. Wecht (0.4 centimeters) and Dr. Green (0.55 centimeters). Furthermore, Dr. Hurwitz noted that the diagnosis of cor pulmonale is not simply pathologic, but rather cor pulmonale is a "clinical pathologic diagnosis." Dr. Hurwitz noted that regardless of the exact measurement, the single measurement does not in itself constitute the basis for a diagnosis of cor pulmonale. Dr. Hurwitz testified that Mr. Clowser had none of the clinical manifestation of cor pulmonale. Dr. Hurwitz explained that, on October 18, 2001 (*i.e.,* less than a week prior to the miner's death), Dr. Rosati's examination "demonstrated jugular venous distension, no peripheral edema. This man clearly did not have cor pulmonale." Moreover, Dr. Hurwitz stated that his opinion regarding the absence of cor pulmonale would not change even assuming Dr. Green's measurement is accurate (EX 6, pp. 18-20). Following his further discussion of the evidence, Dr. Hurwitz reiterated that he finds "no pulmonary component in this man's death. In my opinion this man died of an acute myocardial infarction." (EX 6, p. 32).

Dr. Gregory J. Fino is a B-reader who is Board-certified in Internal Medicine and Pulmonary Disease (EX 2). Dr. Fino issued a report, dated September 9, 2003, in which he reviewed various medical data, including chest x-rays, CT scans, pulmonary function results, cardiovascular stress test, hospital records from October 1995 and October 2001, the miner's death certificate, autopsy report and those by Drs. Brayer and Bush. In addition, Dr. Fino reported a work history of "hauling coal for 35 years" and conflicting smoking histories which, "at either end of this range would cause a significant lung problem." (EX 2). In summary, Dr. Fino stated, in pertinent part:

> There is no evidence whatsoever that lung disease, regardless of cause, played any role in his death. Based on the medical records, there is evidence of clinical and legal coal workers' pneumoconiosis. However, I believe that the abnormalities that I see in this case in terms of pulmonary function can be explained by smoking. The fixed obstructive abnormality with an elevation in lung volumes and a reduction in diffusing capacity are consistent with smoking. In addition, the amount of pneumoconiosis that was described is insufficient to cause an impairing or disabling pulmonary condition.
>
> He was disabled due to lung disease. However, the disability was due to smoking.

His death was due to heart disease. His lung disease, regardless of etiology, played no role. It did not hasten this man's death. He would have died as and when he did had he never stepped foot in the mines.

Conclusions

1. Simple pneumoconiosis was present.

2. There was a disabling respiratory impairment due to smoking. Pneumoconiosis played no role in this disability.

3. Death was due to coronary artery disease.

4. This man's death was not caused, in whole or in part, by the inhalation of coal mine dust.

5. This man would have died as and when he did had he never stepped foot in the mines. Coal mine dust did not hasten his death.

(EX 2).

Dr. Fino also testified at a deposition held on August 27, 2003 (EX 5). Dr. Fino stated that Mr. Clowser had coal worker's pneumoconiosis at the time of the miner's death based on the autopsy evidence, even though it was not evident clinically during his lifetime (EX 5, pp. 12-15). Furthermore, Dr. Fino expressly addressed Dr. Green's opinion, as follows:

> Doctor Green says that in his opinion that myocardial ischemia and hypoxemia was the immediate cause of death. I agree with that. That means that the heart muscle did not get enough blood and oxygen. I have no problem with that. That's what a cardiac arrest is. He then said reduced oxygen supply to the heart in turn was caused by poor blood profusion, coronary artery atherosclerosis, meaning blockages of the arteries. I agree with that. He wasn't getting enough blood. Anemia, this man had a low blood count. I agree with that.
>
> And then he says, and lowered oxygen transfer from the lungs. Pneumoconiosis and dust-induced COPD. Well, I don't agree with that, because I don't see any evidence that there is a lowered oxygen transfer from the lungs, i.e., low blood gases, low oxygen saturations. That I don't buy. There's no objective evidence of that.
>
> But let's assume hypothetically that there were lowered blood oxygen levels. Well, that does occur in chronic pneumoconiosis and chronic lung disease, but that's just what the studies have addressed in the past, and that does not increase your risk of dying due to coal mine dust related lung disease, or heart disease. You don't die more often from heart disease if you're a miner as compared to not being a miner. So I don't agree with him, and I don't think the objective data supports his belief.

(EX 5, pp. 22-24). Following further discussion of the evidence, Dr. Fino reiterated that even though the miner had a significant lung problem due to smoking, this significant lung problem had no effect on the miner's cardiac condition and death. To the contrary, Dr. Fino testified that Mr. Clowser "would have died as and when he did had he not suffered the lung problem regardless of cause." (EX 5, p. 35).

Dr. Francis H.Y. Green, who is Board-certified in Anatomic Pathology and has published extensively (CX 2), issued a report, dated February 6, 2003 (CX 1). Dr. Green listed the medical data which he reviewed, summarized his findings on examination of the autopsy slides, and reviewed the miner's medical history. Based upon the foregoing, Dr. Green opined that Mr. Clowser has simple pneumoconiosis and that the disease contributed to the miner's death. In reaching these conclusions, Dr. Green stated, in pertinent part:

> The autopsy revealed that Mr. Clowser had evidence of simple coal worker's pneumoconiosis comprising macules, micro- and macronodules and interstitial fibrosis…The largest macronodule measured 0.8 cm in greatest dimension…
>
> Mr. Clowser's work history is somewhat confusing. It is my understanding that he only has 4.5 years of accredited coal mining experience, whereas the clinical record indicates that he may have worked up to 35 years in the coal industry, predominantly hauling coal…
>
> Mr. Clowser's lungs also showed evidence of severe emphysema and chronic bronchitis. These anatomical findings correlate with the clinical history of obstructive lung disease and radiographic evidence of emphysema. Chronic obstructive lung disease due to a combination of emphysema and chronic bronchitis is increased in coal miners…
>
> Mr. Clowser had clinical evidence of pulmonary hypertension (mean pressure of 72 mmHg) based on an echocardiogram of May 18, 1999 (as reported by Dr. Larry Hurwitz, September 3, 2002). At autopsy, the right heart was slightly enlarged, and measurement of the right ventricle on the single identifiable section of the right ventricle at autopsy showed a dimension of 0.55 cm, which is slightly greater than normal (0.3 – 0.4) cm). This in conjunction with sclerosis of the vessels in the lungs at autopsy are consistent with pulmonary hypertension.
>
> Mr. Clowser had a history of cigarette smoking with an approximate cumulative pack-year history of 40-45 pack years. According to the medical records, Mr. Clowser quit smoking in 1994. This smoking history is significant and would also have contributed to Mr. Clowser's chronic obstructive pulmonary disease. Nonetheless, based on the amount of dust in his lungs and the degree of pneumoconiosis, it is my opinion that coal mine dust exposure was a significant causative factor for his COPD.
>
> In summary, Mr. Clowser had pathologic evidence of simple coal worker's pneumoconiosis (including silicosis) together with severe chronic obstructive pulmonary disease, predominantly emphysema; both of these conditions were in whole or in part the

> result of exposure to coal mine dust. These lung diseases were responsible for the clinical and pathologic evidence of pulmonary hypertension (cor pulmonale).
>
> As stated above, it is my opinion that myocardial ischemia/hypoxia was the immediate cause of death. Reduced oxygen supply to the heart in turn was caused by poor blood perfusion (coronary artery atherosclerosis), anemia (myelodysplastic syndrome) and lowered oxygen transfer from the lungs (pneumoconiosis and dust-induced COPD). Without the additional stress of the pneumoconiosis, it is very unlikely that he would have suffered the fatal cardiac arrhythmia and/or infarction. Therefore, it is my opinion, to a reasonable degree of medical certainty, that Mr. Clowser's pneumoconiosis was a direct contributing factor to his death.

(CX 1).

In a supplemental report, dated August 19, 2003 (CX 3), Dr. Green stated that he had obtained the information regarding the May 18, 1999 echocardiogram from Dr. Hurwitz's report, dated September 3, 2002, not his own independent review of the echocardiogram report. Therefore, Dr. Green noted that if there is an error, it lies with Dr. Hurwitz. In addition, Dr. Green discussed the thickness of the right ventricle, in particular the difference between his own finding of 0.55 cm, and Dr. Wecht's measurement of 0.4 cm. Dr. Green noted that Dr. Wecht measured fresh heart specimen, while he measured a fixed and stained section of the right ventricle. In any event, Dr. Green stated that the "differences are not major." He described Dr. Wecht's finding as "the upper limit of normal," and his own as showing "slight hypertrophy." In summary, Dr. Green stated, impertinent part: "Once the clinical data is clarified, I would be glad to modify my report, write an addendum and/or give a deposition." (CX 3).

In deposition testimony on February 11, 2002 (CX 4), Dr. Green stated that he had reviewed various reports relating to Mr. Clowser, including Dr. Tomashefski's report, as well as transcripts of the depositions of Drs. Bush and Hurwitz and other records (CX 4, pp. 6-7). Following his discussion of the evidence, Dr. Green reiterated that Mr. Clowser had pneumoconiosis which caused pulmonary hypertension and a mild degree of cor pulmonale. Furthermore, unlike Dr. Hurwitz, who described cor pulmonale as a clinical pathologic diagnosis, Dr. Green stated that cor pulmonale is defined pathologically (CX 4, p. 23). Moreover, Dr. Green reiterated that pneumoconiosis was a substantial contributing factor in the miner's death. Dr. Green testified, in pertinent part:

> The symptom of angina that he had, which is the clinical evidence that he was suffering low oxygen supply to the heart, would be exacerbated, and I think in a significant way that would contribute to his death because I think the evidence points to the fact that he died suddenly most likely from an arrhythmia. And the major factor causing the low oxygen supply would be the lung disease. So he could well have lived quite a time longer had he not had the lung disease.

(CX 4, p. 36).

Dr. Joseph F. Tomashefski, Jr., who is Board-certified in Anatomic and Clinical Pathology and has published extensively, issued a report, dated December 23, 2003 (EX 3). Dr. Tomashefski listed various medical records which he reviewed. In addition, he reviewed the histologic slides of the miner's heart and lungs. Dr. Tomashefski stated impertinent part:

> Based upon my review of the medical records and the slides of Mr. Clowser's heart and lungs it is my opinion that he had severe coronary atherosclerosis with histological evidence of myocardial fibrosis, organizing microinfarct, and a small intramyocardial coronary thrombus. Within reasonable medical certainty, the immediate cause of Mr. Clowser's death is an acute myocardial infarct. The histological absence of acute myocardial necrosis is due to the short interval between the ischemic event and death. Histological evidence of an acute myocardial infarct usually takes about 6 to 12 hours to become apparent.
>
> It is also my opinion that Mr. Clowser had diffuse, predominantly panacinar emphysema and chronic bronchitis which correlates with the obstructive changes noted on his pulmonary function tests.
>
> Based on the findings of a few coal macules and a single, 0.7 cm macronodule, it is also my opinion, within reasonable medical certainty, that Mr. Clowser had very mild, simple, coalworkers' (sic) pneumoconiosis. Mr. Clowser's simple coalworkers' (sic) pneumoconiosis is of low profusion and would not, in my opinion, have caused him any respiratory symptoms or functional impairment. Within reasonable medical certainty, Mr. Clowser's mild, simple coalworkers' (sic) pneumoconiosis was not a cause or a contributory factor to his death.
>
> It is also my opinion that Mr. Clowser did not have cor pulmonale. He had none of the clinical stigmata of cor pulmonale, his heart was of normal weight, and the thickness of his right ventricle, recorded at autopsy, was normal.
>
> It is also my opinion, within reasonable medical certainty, that neither mild simple coalworkers' (sic) pneumoconiosis, coal dust exposure, nor coal mine employment was a cause or a significant contributory factor in the development of Mr. Clowser's diffuse emphysema. There was no specific spatial relationship between diffuse emphysema and the few small lesions of coalworkers' (sic) pneumoconiosis that were present. If Mr. Clowser's diffuse emphysema had been due to coal dust exposure one would expect to see a much more severe degree of simple coalworkers' (sic) pneumoconiosis. Furthermore, pancinar emphysema is a subtype that is not typically associated with coal dust exposure or coalworkers' (sic) pneumoconiosis. Within reasonable medical certainty, Mr. Clowser's diffuse emphysema was caused by his sustained exposure to cigarette smoke over many years.
>
> In my opinion, Mr. Clowser's death was due to an acute myocardial infarct that was directly related to his severe coronary atherosclerosis, and unrelated to his COPD or mild coalworkers' (sic) pneumoconiosis. A medical examination performed one day prior to

his death indicated that he was not in respiratory distress, and both his lung and cardiac auscultatory examinations were normal.

(EX 3). Following a further discussion of Dr. Green's opinion, and multiple criticisms thereof, Dr. Tomashefski concluded:

> Within reasonable medical certainty, it is my opinion that Mr. Clowser would have died at the same time and in the same manner even if he had never worked as a coal miner or developed mild simple coalworkers' (sic) pneumoconiosis.

(EX 3).

Discussion and Applicable Law

As set forth above, Employer stipulated, and I find, that Mr. Clowser had simple pneumoconiosis. Furthermore, in view of my finding of more than ten years of coal mine employment, the rebuttable presumption that the disease arose from coal mine employment is applicable. 20 C.F.R. § 718.203 and § 718.302. This presumption has not been rebutted. However, in order to be eligible for benefits, Claimant must also establish that the miner's death was due to pneumoconiosis, as provided in the Act and applicable regulations.

Death due to Pneumoconiosis

Since the claim was filed after January 1, 1982, the issue of death due to pneumoconiosis is governed by § 718.205(c), as amended, which states, in pertinent part:

> For the purpose of adjudicating survivor's claims filed on or after January 1, 1982, death will be considered to be due to pneumoconiosis if any of the following criteria is met:
>
> (1) Where competent medical evidence establishes that pneumoconiosis was the cause of the miner's death, or
>
> (2) Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis, or
>
> (3) Where the presumption set forth at § 718.304 is applicable.
>
> (4) However, survivors are not eligible for benefits where the miner's death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death.

(5) Pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death.

20 C.F.R. § 718.205(c).

As outlined above, there is no medical evidence which establishes that pneumoconiosis was the immediate and/or primary cause of the miner's death. Furthermore, Claimant has failed to establish the presence of *complicated* pneumoconiosis. Therefore, I find that Claimant has not established "death due to pneumoconiosis" under § 718.205(c) (1) and (3). Accordingly, the crux of this case is whether or not the miner's found simple coal worker's pneumoconiosis substantially contributed and/or hastened the miner's death. *See* 20 C.F.R. § 718.205(c) (2), (4), (5).

Having weighed the relevant evidence, in particular, the miner's death certificate (DX 11), the autopsy report co-authored by Drs. Omalu and Wecht (DX 12), and the medical opinions of Drs. Bush (DX 19; EX 4), Hurwitz (EX 1, 6), Fino (EX 2, 5), Green (CX 1, 2), and Tomashefski (EX 3), I find that Claimant has not established that pneumoconiosis substantially contributed or hastened her husband's death.

As outlined above, the death certificate, authored by Dr. Brayer, does not even mention pneumoconiosis (DX 11). Although the presence of pneumoconiosis, among numerous other conditions, is cited by Dr. Brayer in a subsequent report, he did not specify that pneumoconiosis caused, substantially contributed or caused the miner's death (DX 18). Similarly, the autopsy report lists pulmonary interstitial anthracosis among numerous diagnoses. However, Drs. Omalu and Wecht did not address the death causation issue (DX 12). Claimant's case rests with Dr. Green's opinion (CX 1-4). Although Dr. Green is a well-credentialed pathologist, his opinion is contrary to those of Dr. Hurwitz, a Board-certified cardiologist, Dr. Fino, a Board-certified pulmonary specialist, and other well-credentialed pathologists (*i.e.,* Drs. Bush, Tomashefski). Furthermore, some of Dr. Green's underlying findings, such as his diagnosis of cor pulmonale, have been rejected by the other physicians as inconsistent with the credible, objective medical data. Moreover, Dr. Green's measurement of the right ventricle is also somewhat inconsistent with the autopsy findings. Even Dr. Green conceded that Dr. Wecht's 0.4 cm measurement represents "the upper limit of normal," and that his own 0.55 cm finding shows only "slight hypertrophy." In addition, this case clearly involves a cardiac death, which Dr. Green stated is causally related to Mr. Clowser's coal mine dust-related pulmonary conditions. However, Dr. Green lacks the credentials of Drs. Hurwitz and Fino, in the relevant fields of cardiology and pulmonary medicine. Drs. Hurwitz and Fino expressly rejected Dr. Green's analysis and his finding of such a causal connection.

In view of the foregoing, I find that the opinions of Drs. Hurwitz and Fino, as buttressed by those of Drs. Bush and Tomashefski, far outweigh that of Dr. Green. Accordingly, I find that Claimant has failed to meet her burden of establishing death due to pneumoconiosis under § 718.205(c), or by any other means.

Conclusion

Although the evidence shows that the miner had simple pneumoconiosis which arose from his approximately 13.5 years of coal mine employment, it does not establish that pneumoconiosis caused, substantially contributed to, or hastened the miner's death. Therefore, I find that the Claimant is not entitled to benefits under the Act and applicable regulations.

Attorney's Fees

The award of an attorney's fee under the Act is permitted only in the cases in which Claimant is found to be entitled to benefits. Since benefits are not awarded in this case, the Act prohibits the charging of any fee to the claimant for services rendered to him in pursuit of this claim.

ORDER

It is ordered that the claim of Marilyn L. Clowser, surviving spouse of Donald E. Clowser, for black lung benefits under the Act is hereby **DENIED.**

A

RICHARD A. MORGAN
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal to the Benefits Review Board within 30 days from the date of this Decision and Order, by filing a notice of appeal with the **Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601.** A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Frances Perkins Building, Room B2117, 200 Constitution Avenue, N.W., Washington, D.C. 20210.